

two in five. What will this tell us respecting the certainty of the next case? We cannot know whether it will be one of the two or one of the three. We ought to know what are the conditions which will range it infallibly under one or the other head. Instead of an idle enumeration, we should make a fruitful study of each special case, and discover, if possible, the cause which renders the operation fatal. The same reasoning applies to curative remedies. A certain remedy has in twenty instances been followed by a cure; in seven instances no cure has been effected. You will say, perhaps, that there is twenty to seven in favour of success. Not in the least. You do not know how many of those twenty patients would have recovered had there been another remedy tried, or no remedy at all; you do not know what was the precise action of the remedy, what changes it effected in the organism, what its effects will be on the organism now about to be submitted to it. As a great mathematician observed, "La loi des grands nombres est toujours vraie en général et fausse en particulier." And as to the "compensations which bring about the law," they are useless in medicine. Mathematicians admit that if a red ball has come up fifty times in succession, that is no reason why the white should come up on the fifty-first; the white ball is certain to come up some time or other, but its appearance depends on specific conditions which have nothing to do with what has gone before.

Is there, then, no utility in statistics? M. Bernard is far from saying so. He admits that statistical results lead to probabilities and suggest research; but he protests against the idea that medicine must be only conjectural. He insists on the necessity for a scientific basis, and declares that every method of treatment which is not grounded on a clear recognition of the causal connections between agents and the organism is mere empiricism, not much removed from charlatanism. "Les médecins en général semblent croire qu'en médecine il y a des lois élastiques et indéterminées. Ce sont là des idées fausses qu'il faut faire disparaître si l'on veut fonder la médecine scientifique. La médecine, en tant que science, a nécessairement des lois qui sont précises et déterminées, qui, comme celles de toutes les sciences, dérivent du critérium expérimental."—*Brit. Medical Journal*, Dec. 16, 1866.

11. *Morbid Anatomy and Early Physical Signs of Pneumonia*.—Dr. T. H. WATERS read (Dec. 12, 1865) a paper on this subject before the Royal Medical and Chirurgical Society.

He states that "although the conditions which characterize pneumonic inflammation—engorgement, red hepatization, and gray hepatization—have been accurately described by various pathologists, discrepancy of opinion exists on some points connected with the morbid anatomy of the disease, such as, 1st, the bloodvessels involved in the inflammation; 2d, the particular part of the pulmonary substance which is the seat of the disease. These points can only be cleared up by a careful comparison of the healthy with the pneumonic lung. With regard to the bloodvessels involved in pneumonia, the opinions of pathologists are divided. Some believe that the capillaries of the pulmonary artery are those essentially affected, those of the bronchial arteries being most probably also involved; some consider the bronchial capillaries mainly concerned; while others think it probable that both sets of vessels are simultaneously affected, although perhaps in different degrees. In considering this question it is necessary to examine into the arrangement of the bloodvessels of the lungs, to define clearly the parts to which each set is distributed, and to ascertain the exact portions of the pulmonary substance involved in the pneumonic inflammation. The true respiratory portion of the lungs consists of a series of air sacs situated at the extremity of each bronchial tube. These air-sacs are separated from each other by thin membranous walls. The pulmonary arteries are the only bloodvessels distributed to the air-sacs. These vessels ramify in the walls of the sacs, and form in them the pulmonary plexus. They are engaged not simply in carrying blood for the special function of the lungs, but also for the nourishment of the tissue to which they are distributed. Although the bronchial arteries pass along the bronchial tubes and supply the structures of those tubes and the areolar tissue of the lungs, they send no branches to the walls of the air-sacs,

which are solely occupied by the plexus formed by the pulmonary artery. In speaking of the areolar tissue of the lungs, the author wished it to be distinctly understood that no such tissue is found in the walls of the air-sacs; it is only demonstrable in the adult lung around the bronchial tubes, the larger bloodvessels, and the lobules, as well as beneath the pleura. Such being the distribution of the bloodvessels of the lungs, and the arrangement of the areolar tissue, the next point for consideration is the exact seat of the pneumonic inflammation. On examining, under the dissecting microscope, a piece of inflamed lung which has reached the stage of hepatization, it is at once seen that the seat of the exudation is the air-sacs. These cavities are filled with solid matter; and, if the preparation has been kept in spirit for some time, moulds of the cavities can be drawn out. As the air-sacs are the seat of the exudation, it is obvious that the exudation must be poured out from their walls. The structures composing these walls must, therefore, be the seat of the inflammatory process; and as they contain no other vessels than those derived from the pulmonary artery, it is the branches of this vessel alone which are involved in the disease. In a piece of hepatized lung, exudation is sometimes found in the smaller bronchial tubes; at other times it is absent from them, and merely fills the air-sacs. Its presence in the bronchial tubes by no means proves that it has been poured out from their lining membrane; for it may have passed into the tubes from the air-sacs, being pressed out from the latter as they have become over-distended. In some cases of pneumonia there is no reddening of the mucous membrane of the finest bronchial tubes—no post-mortem appearances to show that there has been anything more than a simple uncomplicated pneumonia; whilst, in other cases, an increased vascularity of the bronchial membrane indicates the concurrent existence of bronchitic inflammation. Some pathologists, in speaking of the morbid anatomy of pneumonia, have described the exudation as taking place, in part, into the interstitial tissue. They have not, however, described accurately what they mean by interstitial tissue; and it is very important that clear notions should exist with reference to this particular point. The author has already mentioned that the lungs are not permeated throughout with areolar tissue, and that it only exists in certain parts, and in small quantities. The true lung-tissue—that which has been known as the parenchyma of the lung—consists of the walls of the air-sacs. These walls are firm and strong, but very thin. They consist of yellow elastic tissue, and a basement membrane, inclosing the pulmonary plexus. No areolar tissue is found in these walls; a fact which the morbid condition produced by pulmonary emphysema fully demonstrates. Although in pneumonia the walls of the air-sacs become somewhat thickened, this is due, the author believes, chiefly to the enlargement of the capillaries which they contain, and only partly to their retaining some of the exudation. Grisolle thinks that in pneumonia the capillaries are very probably augmented in number. It is impossible to decide positively with reference to this point, but the author's opinion is decidedly opposed to that of Grisolle. He believes that no development of new vessels takes place; they increase in size, but not in number. In gray hepatization, the air-sacs are still the seat of the exudation, and no destruction of their walls takes place unless an abscess is formed. There is no interstitial suppuration; the exudation-matters, in the process of cure, are either reabsorbed or expectorated. The following conclusions are drawn from the foregoing facts: 1. That pneumonia consists of an inflammation of the walls of the air-sacs of the lung. 2. That the bloodvessels involved in pneumonia are the branches of the pulmonary artery, which constitute the pulmonary plexus; and that the capillaries of the bronchial arteries are in nowise implicated, unless there be a concurrent bronchitis, which is an addition to the pneumonia, and not an essential part of it. To pass to the second subject of the paper—the early physical signs of pneumonia. The author believes that engorgement is not the first morbid change that takes place in pneumonia, and agrees with Dr. Stokes that there is a prior stage, characterized by a dryness of the pulmonary membrane, and probably intense arterial injection. In proof of the probability of this condition, an appeal is made to the facts furnished by auscultation—viz., the existence of a dry, harsh, loud, respiratory murmur preceding the crepitating rale. Two cases have lately been under the author's care in which the existence

of a loud respiratory murmur was noted as an initial physical sign of pneumonia. In both cases there was acute primary pneumonia coming on in lungs previously healthy."

Dr. C. J. B. Williams said that some thirty years ago he had worked a good deal on the subject connected with the paper, and he was glad that the author confirmed the principal point with regard to the essential seat of pneumonia which he had propounded so long since. This was, that the inflammation essentially occupies the great pulmonary plexus of vessels; and that its occupation of these vessels, through which all the blood of the body has to pass, gives to pneumonia much of its grave character. In fact, from this circumstance pneumonia becomes much more than a local affection—it partakes of the characters of a blood disease; and its symptoms are more constitutional than those of any other local inflammation. Thus in sthenic the whole body is intensely heated; in typhoid pneumonia the several symptoms are those of typhus or blood-poisoning; in suppurative pneumonia they are those of pyæmia. The important bearing of this on treatment is obvious. He would further remark that other pulmonary diseases besides pneumonia, especially affecting the great pulmonary plexus of vessels, also manifested their effects on the blood of the whole body. Thus in general emphysema of the lungs, which wastes and reduces many of the pulmonary vessels, the system becomes anæmic and cachectic; and in extensive pulmonary tuberculosis the degenerative or scrofulous tendency rapidly pervades the body. So far, then, as Dr. Waters referred the primary seat of pneumonia to the pulmonic capillaries, he (Dr. Williams) agreed with him. But when he went on to exclude the extreme bronchial and other tissues from a share in the lesion, and to deny the very existence of any intervesicular or filamentous tissue, he (Dr. Williams) would wait for a further confirmation before he could accept his view. He had himself ascribed the peculiar rusty, viscid expectoration of pneumonia to an extension of the inflammation to the mucous membrane of the minutest bronchi, and had attributed the fine crepitant rhonchus to the forcible passage of air through this viscid mucus into the cells; and he still doubted that a better explanation could be given. There was something quite characteristic in this sign, consisting of a series of fine, sharp crepitations accompanying inspiration, which seem to be close under the ear, and limited to the spot of vesicular tissue which is their seat; and not, as in the case of bronchial rhonchi, heard in adjoining parts. Its ephemeral duration, too, was significant of the tendency of the inflammation to go on to more complete obstruction and consolidation, which superseded the crepitation by other signs. No doubt, if Dr. Waters's observations were exact, the pathology of pneumonia would be much simplified; but he (Dr. Williams) doubted whether they were compatible with the occurrence of certain varieties of pneumonia which had been described by himself and others, and which seemed to point out a variation in the extent of the inflammation, involving other tissues beyond the mere pulmonary plexus. Neither could he (Dr. Williams) agree with Dr. Waters in the importance which he attached, or the explanation which he gave, to the partial loud respiratory sound which sometimes precedes the crepitant rhonchus. In many cases it is never heard, for the breath sound is weaker than usual before the crepitus begins. In other cases, an irregular, exaggerated sound is sometimes heard; but it appeared to him to be a supplementary movement in the parts around the spreading obstruction, rather than to be due to dryness of the vesicles, as supposed by Dr. Waters. And here he (Dr. Williams) would take occasion to remark, that it is a mistake to suppose that inflammation causes dryness at any stage in serous membranes, or in parenchymata. In mucous membranes there certainly is dryness at any stage, from the interstitial congestion and effusion interfering with the natural mucous secretion; but in serous membranes it was otherwise—increased exudation attends every stage of inflammation. Some authors had erroneously ascribed the friction sound of pleurisy and pericarditis to dryness of the membrane; but he (Dr. Williams) had ascertained by experiments on animals that the friction sound is always due to a disposition of coagulable lymph, sometimes extremely thin; and this, by its adhesiveness, is the cause of the rubbing sound between the serous surfaces. Neither is there any reason for assuming the existence of dryness, in the early stage of inflammation, of the pul-

monary vesicles, which are as simple as serous membranes. In conclusion, Dr. Williams thought they were indebted to Dr. Waters for his researches; but he did not consider them conclusive on this subject, interesting alike to pathology and diagnosis.—*Med. Times and Gaz.*, Dec. 23, 1865.

12. *Certain Forms of Hæmoptysis, unassociated with Pulmonary Tuberculosis.*—Dr. RICHARD PAYNE COTTON draws attention (*Lancet*, Dec. 2, 1866) to what he believes to be a not unfrequent, though little recognized form of non-tubercular hæmoptysis, met with chiefly in the female sex, but sometimes among males, generally in the early period of life. He relates three cases illustrative of this, one of which is the following:—

“A young lady, aged 18, recently arrived from a residence in one of the West India Islands, was supposed to be phthisical. I was requested to see her, and report upon the nature of her disease, about which several very conflicting opinions had already been given. She was anæmic, nervous, and out of health; had a dry cough, but had not become thinner; her catamenia were regular, but scanty; her appetite was capricious; and she had had frequent hæmoptysis, which, there was every reason to believe, did not proceed from either the mouth or fauces. The blood, upon examination, was found to be thin and watery, of a dark colour, free from coagulum, unassociated with either bronchial or salivary secretion, and in general appearance much resembling a mixture of *red-currant jelly and water*. I was informed that this was its general character. Sometimes it had been considerable—as much as half a pint in twenty-four hours; at others, it would not exceed a teaspoonful or two during the same period; sometimes it would be scarcely enough to tinge a pocket-handkerchief, and often it would disappear for days together. This state of things had existed for nearly two years, causing great anxiety to the patient and her friends, from a belief that it was indicative of pulmonary disease. Careful examination of the chest, however, failed to elicit any evidence that such was the case. Rest, change of air, and the tincture of sesquichloride of iron, entirely restored this patient to health. It is now more than three years since I was consulted, and I heard a short time back that the young lady was in perfect health.”

“Other cases of this form of hæmoptysis,” he states, “have fallen under my observation; but I shall not specially refer to them in consequence of not knowing their sequel. At least twelve or thirteen have happened in my own wards at the Hospital for Consumption. Two only of these were males; the rest were females, generally of delicate and nervous appearance, and under the age of thirty. Several had very suspicious symptoms of phthisis; but the physical signs failed to exhibit any evidence of pulmonary tuberculosis, and most of them improved in health under appropriate treatment. In every case the expectoration was of the same general character; sometimes it was mixed more or less with bronchial mucus, slightly tinged perhaps with blood, and sometimes with salivary secretion; but more frequently it was simply watery blood, resembling, as I have described, a mixture of red-currant jelly with water.”

The following are Dr. C.’s conclusions, drawn from his observations of such cases:—

“1st. There is a form of true hæmoptysis in which the expectoration is of a dark colour, and of a more or less watery consistence, bearing a close resemblance to a mixture of red-currant jelly and water.

“2d. That such hæmoptysis is of non-tubercular origin, and may proceed from any part of the gastro-pulmonary mucous membrane.

“3d. That it is attributable to a morbid and fluid condition of the blood, allied, at least in appearance, to that which is met with in purpura and scurvy.”

13. *Snow's Theory of the Causes of Cholera Explained and Illustrated.*—The following able exposition of Dr. Snow’s theory of the propagation of cholera, given in the *Med. Times and Gaz.*, Oct. 1865, by Dr. B. W. Richardson, will be read with interest.

“The first element of the theory is to the effect that cholera, pathologically, is a disease exclusively of the alimentary canal; that the great primary change from healthy to diseased action is at first locally confined to the alimentary tract;